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TO: Alan Held, Esq., DOJ
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FROM: Paul Mushak, Ph.D. Paul Mushak

RE: Independent Scientific Review of (1) the NL-Taracorp/Granite City Superfund Site ROD with Appendices A & B; (2) the NL Responses to the ROD, (4) Relevant Data from the RI/FS document of NL Industries (O'Brien & Gere) and (4) the 1982 Blood Lead Study of the IEPA.

This review of material for the referenced Superfund site includes both general and specific commentary. Review comments are organized by document being reviewed. I attach my Curriculum Vitae (C.V.) and a C.V. summary to copies for Region V and DOJ; they provide scope of expertise being applied to my independent review of this matter. I confine my remarks to the toxicological and public health risk assessment aspects of the ROD and PRP responses.

1. The Record of Decision (ROD)

The central public health question in the Granite city ROD is the level of health protection afforded the affected public by remediation of lead contamination to a level of 500 mg Pb/kg (500 ppm) in residential soil/driveways near the Superfund site. Region V correctly judges that the NL RI/FS risk assessment document shows pervasive lead contamination of soil and other media in nearby residential areas.

In my opinion, the selected clean-up level for soil and related contaminated media of 500 ppm is at best the upper bound or maximum level permitted for adequate prevention of exposure. A level below 500 ppm, i.e., 300-400 ppm based on use of the EPA biokinetic model, would actually be preferable for assuring adequate protection of the more sensitive subsets of the potentially impacted pre-school child population. The scientific evidence behind this conclusion includes:

(a) The present blood lead level now considered the maximum definition of "safe" in the young child and the pregnant woman is around 10 micrograms/deciliter whole blood (a Pb-B at/below 10 ug/dl). This figure represents the informed opinion of lead experts as expressed in

published or imminent U.S. Federal consensus documents.

These include the sign-off report of EPA's outside experts on the Science Advisory Board, who evaluated the EPA's lead criteria document and its several addenda and updates (CASAC, 1990). CASAC explicitly stated that a Pb-B of 10 ug/dl is to be the MAXIMUM safe level (NOT an average Pb-B for a group). These latter EPA documents themselves give a range of 10-15 ug/dl as the threshold for a constellation of early effects, of which the critical, early neurotoxic effects are noted at/below 10 ug/dl (U.S. EPA, 1986; 1989; 1990; Grant and Davis, 1989). The U.S. ATSDR (ATSDR, 1988; Mushak et al., 1989) identified a range of 10-15 ug/dl for multiple effects with selection of 10 ug/dl as the onset for early but important neurobehavioral effects such as an IQ decrement and related neurobehavioral measures. Draft language of CDC's imminent 1991 Statement on childhood lead poisoning defines 10 ug/dl as the earliest action level for lead toxicity (NY Times, 12/20/90). Consequently, health risk assessment in the ROD, responses to the ROD and critiques of related material must all be based on a Pb-B level of 10 ug/dl.

(b) The ROD and associated material such as NL's RI/FS document do not qualitatively or quantitatively consider the reasonable maximum exposure (RME) in addition to average exposure scenarios. Such a deficiency not only understates public health risk for a segment of risk children who have elevated intakes and uptakes of lead but appears to go against what is present EPA policy towards Superfund sites, i.e., assessment of both average exposures and RMEs. If a soil clean up level of 500 ppm is required based on average intake assumptions for soil, dust, vegetables, etc. then a lower soil level would be required for children having higher intakes. An RME would include intake rates at the 95%-ile and lead in soil and vegetables at the 95%-ile.

(c) The use of EPA's validated Integrated Uptake/Biokinetic (IUBK) Model for the Granite City site is certainly appropriate, considering site-specific lead levels from the RI/FS document, for estimating Pb-B levels as a function of different soil lead clean-up guidelines. The IUBK model approach is a flexible means of estimating resulting blood leads under a series of exposures scenarios that are not constrained by an existing set of circumstances. It can be used for average exposures and RMEs and for present vs. future land-use scenarios. Such flexibility is not possible in a specific Pb-B survey at a fixed point in time.

Appendix B, Fig. 1 of the ROD shows that a rather high, arguably unacceptable fraction of children would have a blood lead above 15 ug/dl at a cut-off level of 500 ppm soil lead. The use of a Pb-B level of 10 ug/dl (see above) would produce an even higher fraction of children at an elevated toxicity risk (> 10 ug/dl). Levels of 300-400 ppm

lead as the selected level would of course reduce this fraction sonsiderably.

(d) Appendices A and B are deficient in not using more recent soil lead-blood lead studies and relevant consensus documents to make their case, although the evidence used collectively in the ROD makes an acceptable argument for selection of a soil lead level of 500 ppm. In some cases, the authors of the ROD should have looked at studies at certain other Superfund sites relevant to the matter, e.g., the Bunker Hill, ID site (EPA, 1989a). I note below empirical data that support a clean-up level of 500 ppm as the maximum.

- A joint study by the Colorado Department of Health, ATSDR and the University of Colorado at Denver (CDH, 1990) was carried out at a smelter-mill site in Leadville, CO. Blood lead and environmental lead data were gathered using a very rigorous epidmiological design and soil Pb-blood Pb relationships were analyzed using multiple regression analysis and the "odds ratio" approach for soil Pb and Pb-B relationships. This well-done study found that blood lead rises at a soil Pb level of 500 ppm and higher. Since this site would have had lead in forms probably somewhat less bioavailable than at Granite City, the 500 ppm threshold would be an upper bound or maximum for Granite City.

- The Granite City site is also best compared as to exposure criteria to a Midwestern site with many aspects of exposure similarity, i.e., the Omaha, NE area as studied by Angle and co-owrkers (Angle et al., 1984). The Omaha, NE studies showed that blood lead rises about 7 ug/dl for each 1000 ppm lead in soil and/or dust. At a selected clean-up level of 500 pm for Granite City, this means 3.5 ug/dl blood lead would be permitted. Compared to a guideline value of 10 ug/dl (see above), this adds a sizeable 35% to the total.

In summary, current scientific evidence clearly shows that a soil lead level of 500 ppm at a Superfund site with Granite City characteristics is, at best, the maximum level that could be permitted in a clean-up. Use of a prudent RME approach in tandem with use of a blood lead cut-off of 10 ug/dl for the biokinetic model Pb-B distribution in Appendix B of the ROD would however argue that a level of 500 ppm is actually too high for adequate health protection of a certain fraction of lead-exposed children. The studies used in deriving values selected in the ROD for clean-up collectively support a cut-off level for remediation of 500 ppm lead in soil although, again, there are much more data that could have been included. Additional comments directed to the ROD are given below in my critique of NL Industry's responses to the ROD and other documents.

2. NL Industries' Criticisms of the ROD

My comments about NL Industries' responses to the ROD are based on the totality of scientific evidence. I do not confine my comments only to specific studies used by NL in their criticisms.

GENERAL COMMENTS

In general, I find the NL Industries' public responses document for the subject ROD (hereafter NL document) to be relatively uninformed in a number of places and also misleading in others. This is both with regard to the body of evidence used by EPA to issue the ROD and the overall available scientific data on soil Pb vs. lead toxicity risk.

Specifically, the arguments of NL Industries for a soil clean-up level of 1000 ppm lead at the NL/Taracorp site in Granite City are unsupported by either site-specific data or the general array of scientific evidence. There are a number of assertions in the NL document which charge that the ROD lacked adherence to EPA guidance in decision-making. Whatever the scientific virtue of all such arguments, one can argue that the ROD should have seriously considered the reasonable maximum exposure scenario as the guiding principle for identification of a lead soil level for clean-up. This would have had, however, the opposite effect to that desired by NL Industries.

The NL document is strong in supporting the PRP's RI/FS document prepared by O'Brien and Gere, not surprising since authors of the latter are included as authors of the former. There are however a number of shortcomings of the RI/FS study a I note these later. The assertion in the NL document that the Illinois EPA has not been a partner in this ROD is not a scientific matter. I would note that Illinois has signed off on the Alternative H selected in the ROD.

The NL document draws upon the 1982 IEPA blood lead study to argue that there is no imminent or substantial endangerment at this site. This Pb-B study is not scientifically valid support for any such argument, given the many problems with this study. I critique this study in some detail later in my comments.

In overview, the scientific and public health evidence for the use of a soil clean-up level of AT LEAST 500 ppm lead is compelling and use of such a level by EPA is not scientifically arbitrary and capricious vis-a-vis a higher lead clean-up level. If anything, the available evidence arguably supports a clean-up level below 500 ppm, 300-400 ppm.

SPECIFIC COMMENTS

The use of a reference dose/ADI argument for lead is now obsolete, is not in accord with guidance and is scientifically indefensible, since it is the 2-year old child who is the risk subject to be protected from neurotoxic injury at Superfund

sites, using at minimum the biokinetic model approach to estimate body burdens of lead accruable from site-spcific lead loadings of contact media. The inapplicability of the lifetime ADI approach for lead probably pre-dates the preparation of the NL document.

The NL document argues that the IEPA blood lead study in November and December of 1982 does not pose problems in terms of understating the level of Pb-B which would have been found in the summer. The blood lead of children is a very transitory index of exposure if presented as a single measure at a contamination site, reflecting recent exposures over the past month or six weeks. The "half-life" of a given elevation of Pb-B is ca. 30-45 days. The evidence for this is substantial.

The NL document selected a single study, the Cincinnati prospective lead study, to show data for a much longer half-life of 10 months. This half-life is quite untypical, reflecting an unusual case of children with in-utero lead accumulation carrying over to the post-natal period coupled with continuing high levels of exposure in the high-lead environment of these inner-city low income children. They are also not shown to be demographically or toxicologically typical of Granite City children. The NL document authors either are ungrounded in lead toxicokinetics or are trying to define exceptions as the rule. In brief, one does not do a blood lead survey in November/December in a cold part of the country and expect any informed scientist to believe it reflects children's lead exposure during summer play in contaminated soil.

The NL document argues that NL's selection of 1000 ppm as clean-up level for soil lead is based on more scientifically current and rigid studies. This is not in fact the case, in so far as the various studies that are available for assessment do not support a 1000 ppm figure. The authors seem to be unaware of all of the scientific specifics of the studies they selected for support of their arguments.

First, the document inaccurately depicts the intent of the discussion on slope values in the 1986 EPA lead criteria document, Chapter 11. The discussion in EPA's document was to inform the policy maker of the range of slope values for Pb-B vs. soil/dust Pb (per 1000 ppm). it was not to define or mandate what ought to be a single slope value for all cases. In fact, the high range in slopes discussed at length in the EPA document argues that a given site under discussion ought to be matched up with published sites that are closest in all characteristics. As a principal co-author of EPA's four-volume lead criteria document I can speak to the intent of language in this criteria document.

The Stark et al. 1982 study is not relevant to the site-specific characteristics of the Granite Sity site. The Stark study is based on New Haven, CT's inner city neighborhoods in which auto lead fallout, level of colder weather, relative impact of paint lead ingestion over soil lead, etc. are markedly different parameters.

The Rabinowitz and Bellinger, 1988 study appears to be both misunderstood by the authors. It is also a study arguably distinct from Granite City, in terms fo demographic and socio-economic profiles. First, the overall slope in this study is composed of segments which differ in value. The slope at the lower, more relevant end of the soil range is much higher and more rectilinear than the overall curvilinear slope that includes the total soil range. This is typical of the phenomenon of (1) curvilinear response of blood lead to media lead over a huge range but (2) a linear high-slope response at lower, more relevant levels for most cases. This is extensively discussed in chapter 11 of EPA's criteria document. This study would be good for arguing use of a high slope value for the soil range of interest to Granite City.

The Johnson and Wijnburg study, 1988 is in fact a study of a mining and primary smelter area in which one might expect a lower overall relative bioavailability of lead compared to chemical/geochemical lead forms at the Granite City site. Bioavailability is understandably of some interest and concern to the overall issue.

The NL document notes EPA estimates showing that the background blood lead in the nation's children is falling. They imply that this decline essentially allows more of a window for soil/dust lead exposure. A number of misimpressions seem to be held by the NL document authors in this matter of nation-wide trends. I comment on this matter as the senior author of the ATSDR report to Congress which discussed at length th use of national trends and estimates of lead exposure (U.S. ATSDR, 1988; Crocetti, Mushak and Schwartz, 1990a, 1990b).

First, it is statistically invalid to disaggregate national estimates of blood leads in children to describe individual communities; the impermissibility of this is stated in the ATSDR report to Congress. Secondly, overall national blood lead estimates are in fact an aggregate of numerous socio-economic and demographic strata. Urban, industrial areas show considerably higher average Pb-B and prevalences of levels above toxic guidelines than do overall national numbers.

Much is made of the role of lead-contaminated produce grown around the Granite City site in risk assessment and selection of a soil clean-up level of 500 ppm lead. Vegetable contamination is a function of the bioavailability of lead via the soluble fraction of soil lead at the root-soil interface. There is every reason to expect that bioavailability of lead in residential/garden soils around this site would be adequate to produce considerable crop uptake. The chemical forms expected from battery lead smelting and reclamation are bioavailable in soil (see, e.g., the 1986 EPA lead criteria document) and we have the added factor of acidic precipitation (see RI/FS document) helping mobilize soil lead.

The most quantitative overview of this exposure pathway is given in the calculations of Baes et al. (1984). These data indicate that soil with bioavailable lead can contribute to potentially worrisome levels of lead, especially with reference to a current blood lead guideline of 10 ug/dl. Such appears to have been borne out in the actual field data for the Bunker Hill, ID smelter/ mine site that indicated significant intakes of lead (EPA, 1989a).

The NL document criticizes the use in the ROD of data from the Cincinnati lead abatement project as inappropriate. This is not a valid criticism since the Cincinnati project is now yielding one of the better data sets for the purpose of helping establish soil/dust lead impacts on children's body lead burdens. The study is a tightly controlled one, scientifically, with rigorous QA/QC requirements and involves a large research group which has long been active in various aspects of lead research. There is nothing in the Cincinnati plan that precludes its use for Region V's ROD and much to recommend it.

The NL document criticizes the Madhavan et al. study's use of 10 ug/dl as a point of reference. The authors (NL) claim that the effect at issue is simply inhibition of ALA dehydratase, a claimed trivial response. In point of fact, there are a number of neurotoxic effects seen around 10 ug/dl, including IQ decrements, loss of hearing acuity, growth impairment, prenatal toxicity, etc. These results are in Federal peer-reviewed consensus documents mentioned earlier.

3. The O'Brien-Gere RI/FS Document

This reviewer has been involved in the critical review of many risk assessment documents for Superfund sites. From this perspective of experience and my extensive expertise in the lead problem, I conclude that this report is a poor effort in a number of respects.

First, there is a lack of backgrounding and coherence to this report in terms of both scientific/public health issues and EPA Superfund guidance.

Section 8, Risk Assessment, is deficient in its use of out-of-date science even for the year of preparation, 1988. For example, there is indiscriminate mixing of older effect threshold data, Pb-Bs of 30-50 ug/dl, with newer information showing effects at 10-15 ug/dl and perhaps lower. There is a strange reference to a Pb-B of 15 ug/dl being a planned new CDC level for concern; where did that come from?! The treatment of experimental lead carcinogenesis is quite inadequate- the evidence for kidney tumorigenesis in animals induced by divalent lead salts is quite strong and EPA has examined the issue of lead carcinogenesis in a 1989 monograph.

The RI/FS document is deficient in its risk assessment restriction to solely average exposures, i.e., average

concentrations of lead in soil, crops, etc. and an average intake rate in children, rather than inclusion of a higher risk segment of individuals who have exposures above the mean and whose intakes via such as soil lead ingestion are similarly above average. Selection of intake factors, especially, seem to be neglectful of the likely range of lead intakes. The upper end of soil lead ingestion by children is demonstrably above 100 mg/day for non-pica subjects and likely to be 1000 mg and above for those children with excessive ingestion rates.

The RI/FS study seems to place heavy reliance on the quite inadequate IEPA blood lead survey. The IEPA apparently had its own problem with the blood lead survey, since it never issued the results as a formally released, official report. We are left with looking at an interim draft of unknown citation status.

The RI/FS study relies heavily on what has now been abandoned by EPA, i.e., use of the lifetime chronic ADI for lead for risk assessment. EPA gave up on this methodology since this approach failed to adequately take account of the 2 year-old child as the principal risk group and failed to assess comparative lead toxicokinetics, children vs. adults. As the ROD appendix material correctly notes, the old ADI approach for lead understated risk for lead because the adult segment of the age band inappropriately weighted downward the risk during a child's pre-school years of, for example, a huge ingestion rate relative to adults (100 mg/day vs.10 mg/day) and higher intakes generally as a function of body mass.

The RI/FS document, in relating levels of soil lead to blood lead values to obtain slope coefficients selected, for some strange, unexplained reason, the Stark et al. 1982 study. A more appropriate and scientifically valid comparison would be one that matches communities in terms of community similarity and sources of lead exposure. These comparison criteria strongly point one to the Omaha, NE area and the studies of Prof. Angle, MD and her colleagues at the University of Nebraska. As noted earlier, the coefficients for soil and dust lead vs. Pb-B in the Angle et al. 1984 report are ca. 7.0 ug/dl/1000 ppm lead.

4. The 1982 IEPA Blood Lead Survey

This study has too many limitations to be of much use for risk assessment at the Granite city site. First, the epidemiological design in terms of random sampling and sample size is too deficient and there are no data linking blood leads to soil lead levels in a statistically meaningful way using time-concordant data, i.e., blood lead and soil/dust lead data being gathered within the same study.

Too few children were in this survey to make it of much use for the RI/FS document, specifically with reference to statistical sub-grouping of the 46 children by distance of residential area from the NL/Taracorp site.

Children in this survey were not characterized in terms of their lead exposure sources, so that what we have is simply a collection of blood leads in isolation.

The time of the survey was unfortunate. It is the summer when Pb-B levels should be determined owing to such factors as maximal contact time of children with outside soil during extended outside play. Numerous studies document that summer is the period of maximum blood lead levels, leading lead poisoning to be called the "summer disease" as noted in EPA's 1986 lead criteria document. It has also been found that blood lead is lowest around late fall/winter. In colder areas of the country, such as Granite City, we would expect that there would be abrupt reduction in soil exposure with the onset of fall weather and a concomitant marked reduction in blood lead levels.

There are no data available to indicate what was the level of quality for the QA/QC protocol concerning sample collection, transmission, storage and laboratory lead measurement.

Description of the overall results as showing no risk of toxicity is meaningless in terms of current realities for population toxicology associated with lead exposures. Not only is the blood lead level of concern no longer 25 ug/dl, but the original study characterized health risk in terms of both blood lead and elevated erythrocyte protoporphyrin (EP) concentrations. The present guideline uses a blood level of ca. 10 ug/dl without reference to EP.

CITED REFERENCES

Angle, C.R., Marcus, A., Cheng, I.-H. and Stohs, S. J. 1984. Omaha childhood blood lead and environmental lead: A linear total exposure model. Environ. Res. 35: 160-170.

Baes, C.F., III, Sharp, R.D., Sjoreen, A.L. and Shore, R.W. 1984. A review and analysis of parameters for assessing transport of environmentally released radionuclides through agriculture. U.S. Department of Energy, Oak Ridge National Laboratory, ORNL-5786. September, Washington, D.C.

Clean Air Scientific Advisory Committee Report. 1990. Review of the OAQPS Lead Staff Paper and the ECAO Air Quality Criteria Document Supplement. EPA-SAB-CASAC-90-002. January. Washington, D.C.

Colorado Department of Health. 1990. Leadville Metals Exposure Study. Division of Disease Control and Environmental Epidemiology with University of Colorado Center for Environmental Sciences and Agency for Toxic Substances and Disease Registry. April. Denver, CO.

Crocetti, A.F., Mushak, P. and Schwartz, J. 1990. Determination of numbers of lead-exposed U.S. children by areas of the United States: An integrated summary of a report to the U.S. Congress on childhood lead poisoning. Environ. Health Perspect. 89: 109-120.

Crocetti, A.F., Mushak, P. and Schwartz, J. 1990. Determination of numbers of women of child-bearing age and pregnant women by areas of the United States: An integrated summary of a report to the U.S. Congress on childhood lead poisoning. Environ. Health Perspect. 89: 121-124.

Grant, L.D. and Davis, J.M. 1989. Effects of low-level lead exposure on paediatric neurobehavioral development: Current findings and future directions. Pp. 49-115 in Lead Exposure and Child Development: An International Assessment. M.A. Smith, L.D. Grant and A.I. Sors, Eds., Kluwer Academic Publishers, Dordrecht, Netherlands.

Johnson, T. and Wijnberg, L. 1988. Statistical analysis of lead exposure data collected in East Helena, Montana: Draft Report. January. PEI Associates, Durham, N.C.

Madhavan, S., Rosenman, K.D. and Shehata, T. 1989. Lead in soil: recommended maximum permissible levels. Environ. Res. 49: 136-142.

Mushak, P., Davis, J.M., Crocetti, A.F. and Grant, L.D. 1989. Prenatal and postnatal effects of low-level lead exposure: Integrated summary of a report to the U.S. Congress on childhood lead poisoning. Environ. Res. 50: 11-26.

Rabinowitz, M.B. and Bellinger, D. 1988. Soil lead-blood lead relationship among Boston children. Bull. environ. Contam. Toxicol. 41: 791-797.

Stark, A.D., Quah, R.F., Meigs, J.W. and Delouise, E.R. 1982. The relationship of environmental lead to blood lead levels in children. Environ. Res. 27: 373-383.

U.S. Environmental Protection Agency. 1986. Air Quality Criteria for Lead. Vol. 3 & 4, Ch. 10, 11, 12 and 13. Report No. 600/8-83-028, af-df. Environmental Criteria and Assessment Office, Research Triangle Park, N.C.

U.S. Environmental Protection Agency. 1989. Supplement to the 1986 Lead Criteria Document. March. Environmental Criteria and Assessment Office, Research Triangle Park, N.C.

U.S. Environmental Protection Agency. 1989a. Human Health Risk Assessment Protocol for the Populated Areas of the Bunker Hill Superfund Site. September. Region X Technical Enforcement Support IV Contract, Contract No. 68-01-7351. Seattle, WA.

U.S. Environmental Protection Agency. 1990. Air Quality Criteria for Lead: Supplement to the 1986 Addendum. August. Report No. EPA/600/8-89/049F. Environmental Criteria and Assessment Office, Research Triangle Park, N.C.